

Editorial Commentary

Cognitive processing in migraine

In 1995, Wray, Mijović-Prelac and Kosslyn (1) reported that responses to visual tasks which primarily involved simple feature detection were faster in migraine-with-aura subjects than in controls. In contrast, migraine sufferers did not have a reaction time advantage in tasks that required a higher level of visual processing. Wray et al. concluded that migraine sufferers process signals more rapidly than is normal in the primary visual cortex, the probable origin of transient visual disturbances in the migraine aura. They suggested that cortical ischemia during the migraine aura might selectively damage inhibitory circuitry in the primary visual cortex or, alternatively, migraine sufferers might inherit an unusually excitable visual cortex.

To distinguish between these possibilities, Palmer and Chronicle repeated Wray's experiment with the inclusion of a migraine-without-aura group; however, as reported in this issue of *Cephalalgia*, they were unable to replicate Wray's findings either in migraine-with-aura or migraine-without-aura subjects. It seems unlikely that methodological differences between the two studies account for this discrepancy, because Palmer and Chronicle carefully reproduced Wray's procedures. Perhaps sampling differences were responsible: 5 of the 12 subjects in Wray's sample had migraine equivalents, an unusually high representation; 9 of the 24 subjects in Palmer and Chronicle's sample were studied within 8 days of an attack, whereas Wray's subjects were all studied at least 8 days after an attack. In any event, Palmer and Chronicle's findings indicate that any processing advantage in migraine-with-aura subjects to simple visual stimuli is easily hidden by minor procedural or sampling variations. Since attentional and motor processes have a major influence on response times, measuring the speed of reactions to visual stimuli is only an approximate indicator of visual processing. More specific measures such as visual evoked potentials (2) and event-related potentials (3, 4) have identified abnormalities in vis-

ual processing in migraine sufferers, which, ironically, is more consistent with Wray's data than with the findings presented by Palmer and Chronicle.

A substantial amount of evidence points to visual disturbances which persist subclinically in various guises between attacks of migraine, and then intensify during attacks (5). We still do not know whether these visual disturbances are a fundamental part of the migraine predisposition, or whether they develop secondarily after repeated episodes of migraine. Research into this issue might provide substantial insight into the pathogenesis of migraine. For example, persistent activation of inherently excitable subcortical or cortical neuronal circuitry might eventually recruit a brainstem disturbance that instigates attacks of migraine.

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PD DRUMMOND

Myogenic cerebrovascular autoregulation in migraine

The relevance of new approaches to the puzzling field of migraine pathogenesis is increasing. At this time, transcranial Doppler techniques are ideal for studying "in vivo" cerebral vessel reactivity in migraine patients. Assuming that vascular phenomena

represent part of the headache pathogenesis, it is interesting to know how cerebral vessels react to stereotyped stimuli such as standardized stress (e.g., physical, psychological, etc.). The paper of Heckmann et al., "Myogenic cerebrovascular autoregulation in migraine", demonstrates that there is a different pattern of response to physical stress in terms of cardiovascular resistance indices between healthy subjects and migraine with and without aura. This they attribute to a myogenic mechanism, although a similar mechanism could be preceded by a brief vascular expansion to transiently reduce vascular resistance. It is likely that the initial component of the resistance index reduction is mediated by sympathetic constrictor influences, subsequently sustained by metabolic and myogenic factors. This integrated approach to the study of cerebral vasoregulatory mechanisms should bring new evidence to bear on migraine pathophysiology.

G MICIELI

Hypnic headaches

In 1988, Neil Raskin reported six patients between the ages of 65 and 77 with a previously undescribed sleep-related headache syndrome. The patients awakened from sleep, at a consistent time of night, with a diffuse, variably pulsatile headache lasting between 30 and 60 min. They all responded dramatically to lithium, and three of the six patients were able to discontinue the lithium without headache recurrence. Raskin coined the term, "hypnic headaches" to describe the syndrome. Thereafter, according to the article by Morales-Asin et al. in this issue, there were only two additional patients reported until 1997, a year in which there were reports of 27 new cases. To these, Morales-Asin et al. add three of their own and, also in this issue, Dodick et al. report 19 patients from the Mayo Clinic. These 22 new cases of hypnic headaches expand the boundaries of the syndrome to include: a younger age of onset (40), bilateral or unilateral pain of pulsating or non-pulsating quality, duration up to several hours, occasional spontaneous remis-

sion, and overlapping features with cluster and migraine. Moreover, we now know of two prophylactic agents, other than lithium, that can be given at bedtime to prevent hypnic headaches: flunarizine and caffeine.

RB DAROFF

The second case of chronic paroxysmal hemicrania-tic syndrome

The possibility of two such rare diseases as trigeminal neuralgia and chronic paroxysmal neuralgia occurring ipsilaterally in the same patient, and, furthermore, being diagnosed as such, seems small. Two patients suffering from trigeminal neuralgia-like and chronic paroxysmal hemicrania-like attacks (CPH-tic

syndrome) have now been reported. In the first case, the second and third trigeminal branches were involved and different types of attack occurred separately. The new case first had an episode of V2 trigeminal neuralgia and later a period with V1 trigeminal neuralgia-like attacks combined with CPH-like attacks; the two types of attack were linked to each other.

In the first case, carbamazepine and indomethacin had to be administered to control both types of attack. Carbamazepine did not help the second patient when she had the V2 trigeminal neuralgia, but both types of attack ceased with indomethacin. The medication was stopped after a month without recurrence.

The new case suffered from episodic paroxysmal hemicrania-tic syndrome rather than CPH-tic syndrome.

Because theoretically there are 56 possible variations of this syndrome as to episodic-chronic, concurrent-nonconcurrent, variations as to V1-V3-involvements, and indomethacin sensitive-nonsensitive cases, it is wise to stick to the term CPH-tic syndrome in analogy with cluster-tic syndrome for the moment. Even more variations are possible if the trigeminal neuralgia-like and CPH-like attacks occur contralaterally in future cases. The ipsilaterality of the attacks both in cluster-tic and CPH-tic attacks in all cases so far reported is of great interest, as stressed in the present article. Future reports of similar patients may be of importance for the understanding of the pathophysiology of the symptoms in these different kinds of attacks.

J HANNERZ